CARDIOVASCULAR ISSUES AFTER SPINAL CORD INJURY

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Cardiovascular dysfunction is an important cause of morbidity in SCI, and is one of the two top causes of death in SCI.

1. Cardiovascular Function in SCI

- The parasympathetic supply via the vagus (cranial nerve X) is spared in most SCI. Sympathetic outflow occurs below the T6 level. Injuries above this level generally result in loss of input to sympathetic neurons. So autonomic imbalance occurs in injuries at or above T6 with significant alterations in cardiovascular system control.
- In addition, there are indirect effects of immobility and other SCI related conditions on cardiovascular function.

2. Cardiovascular Concerns in SCI - Occur throughout the care continuum

- **Acute**
  Arrhythmias, bradycardia, cardiac arrest
- **Early, but could be a continuing concern**
  Orthostatic hypotension
  Venous thrombo-embolism
- **Ongoing issues**
  Autonomic dysreflexia
  Reduced cardiovascular fitness
  Cardiovascular effects of medications in SCI
- **Cardiovascular co-morbidities with aging**
  Influence on coronary artery disease
  Peripheral vascular disease

3. Cardiac Arrhythmias

- Arrhythmias are predominantly a concern in the first few weeks after injury, especially bradycardia and even cardiac arrest due to unopposed vagal tone. May be exacerbated by suctioning. Treatment may include atropine, occasionally a pacemaker.
- Arrhythmias do not seem to play a significant role in persons with chronic SCI.

4. Orthostatic Hypotension and Low Baseline Blood Pressure

- Orthostatic hypotension (OH) can occur with acute or chronic cervical/ high thoracic SCI due to altered sympathetic response to posture changes, decreased vascular tone, and decreased venous return. It is more likely to occur with complete injuries.
- **Symptoms and signs**- can include dizziness, pallor, blurred vision, excess sweating above the level of injury, fainting during position change. Symptoms may correlate with altered cerebral blood flow vs. the absolute blood pressure and often diminish with time despite continued evidence of decreased blood pressure.
  - Symptoms may be worse after eating due to post-prandial shunting of blood to the digestive system, in hot environment or after heavy physical exertion, and after prolonged recumbency. Maybe exacerbated by side effect of medications, sepsis, dehydration
  - Post-traumatic syringomyelia may sometimes present with worsening OH

Management

- Non-pharmacological

- Review medications for hypotensive side-effects; minimize exacerbating factors
- Gradual sitting from the recumbent position; progressive postural challenges
- Elevated leg rests; reclining or tilt-in-space wheelchair
- Compression stockings, abdominal binder
- Increased salt and fluid intake if indicated

- **Medications**:
  - **Fludrocortisone acetate** (Florinef)
    Dose: 0.1 -0.4 mg
    Mechanism: Salt and water retention/ delayed action
    Side effects: HTN, CHF, hypokalemia, hypernatremia, weight gain, edema
  - **Ephedrine sulphate** (Ephedrine)
    Dose: 25 mg bid - 50 mg tid, can be given prn prophylactically
    (e.g 20-30 minutes before arising in AM, or before therapy)
    Mechanism: Alpha & beta sympathetic agonist
    Side effects: Tachycardia, arrhythmias, HTN, palpitations, urine retention, insomnia, sweating, tremors, tachyphylaxis, abuse potential
  - **Midoridine hcl** (ProAmitine)
    Dose: 2.5 mg tid - 10 mg tid, avoid giving after 5PM to avoid supine HTN
    Mechanism: Alpha-1 adrenoreceptor agonist
    Side effects: Headache, paresthesias, dysuria, pruritis, piloerection, supine HTN

5. <u>Venous Thrombo-embolism</u>

- The incidence of deep venous thrombosis (DVT) is maximal in the first few weeks after SCI.
- **See the Consortium for Spinal Cord Medicine's Clinical Practice Guidelines on "Prevention of Thromboembolism in Spinal Cord Injury" for detailed recommendations.** In brief, recommendations include:
  - Compression hose or pneumatic devices should be applied to the legs of all patients for the first two weeks following injury.
  - Anticoagulant prophylaxis with either low-molecular weight heparin or adjusted dose unfractionated heparin should be initiated within 72 hours after SCI, provided there is no active bleeding or coagulopathy.
  - The duration of anticoagulant prophylaxis should be individualized depending on the need, medical condition, functional status, and the risk to the patient. Anticoagulants should be continued until discharge in patients with incomplete injuries, for 8 weeks in patients with uncomplicated complete motor injury, and for 12 weeks or until discharge for those with complete motor injury and other risk factors (e.g. lower limb fractures, history of thrombosis, cancer, heart failure, obesity, or age over 70).
  - Vena cava filter placement is indicated in patients with SCI who have failed anticoagulants prophylaxis or who have a contraindication to anticoagulation (such as CNS or GI bleeding). Filters should also be considered in patients with complete motor paralysis due to high cervical cord injury (C2, C3), or with poor cardiopulmonary reserve. However, filter placement is not a substitute for thromboprophylaxis.
- In symptomatic patients perform ultrasound of legs and/or ventilation/perfusion lung scanning. If clinical suspicion is strong but tests are negative or indeterminate, consider venography of the legs, lung spiral CT, or pulmonary angiography.
- With documented DVT, mobilization and exercise of the lower extremity should be withheld 48-72 hours until appropriate medical therapy is implemented.
- Risk for thrombo-embolism is much lower in chronic SCI, and may not be significantly increased after the first year of injury compared to the general population. However,

reinstitution of prophylactic measures should be considered in chronic SCI patients if they have other risk factors (e.g. previous DVT), are immobilized for a long time, or undergo surgical procedures that increase risk for development of DVT in any patient.

6. <u>Autonomic Dysreflexia</u>:

- Results from noxious stimuli which in turn trigger sympathetic hyperactivity. Inhibitory impulses that arise above the level of injury are blocked so that there is unopposed sympathetic outflow. Denervation hypersenstivity of peripheral adrenergic receptors below the level of injry may also contribute to the pathophysiology.
- SCI at the thoracic level T6 or above, i.e. above the major splanchnic outflow, predisposes to autonomic dysreflexia (AD), though occurrence with injuries as low as T8 is described.
- The feature of most concern with autonomic dysreflexia is the significant and potentially life-threatening elevation in blood pressure.
- Recognize the **signs and symptoms** of autonomic dysreflexia, including:
    - Sudden, significant increase in blood pressure.
    - Pounding headache
    - Bradycardia (may be a relative slowing so that the heart rate is still within the normal range)
    - Profuse sweating above the level of the lesion, especially in the face, neck, and shoulders, or possibly below the level of the lesion.
    - Piloerection or goose bumps above or possibly below the level of the lesion
    - Cardiac arrhythmias, atrial fibrillation, premature ventricular contractions, and atrioventricular conduction abnormalities
    - Flushing of the skin above the level of the lesion, especially in the face, neck, and shoulders, or possibly below the level of lesion.
    - Blurred vision
    - Appearance of spots in the patient's visual fields
    - Nasal congestion.
    - Feelings of apprehension or anxiety over an impending physical problem
    - Minimal or no symptoms, despite a significantly elevated blood pressure (silent autonomic dysreflexia).

- **Management**: The Consortium for Spinal Cord Medicine has published clinical practice guidelines for the acute management of autonomic dysreflexia. The summary of these treatment guidelines is as follows[1]:

- Check the individual's blood pressure.
- If the blood pressure is elevated and the individual is supine, immediately sit the person up.
- Loosen any clothing or constrictive devices.
- Monitor the blood pressure and pulse frequently.
- Quickly survey the individual for the instigating causes, beginning with the urinary system.
- If an indwelling urinary catheter is not in place, catheterize the individual.
- Prior to inserting the catheter, instill 2 percent lidocaine jelly (if readily available) into the urethra and wait several minutes.
- If the individual has an indwelling urinary catheter, check the system along its entire length for kinks, folds, constrictions, or obstructions and for correct placement of the indwelling catheter. If a problem is found, correct it immediately.

[1] From the Consortium for Spinal Cord Medicine Clinical Practice Guidelines on Acute Management of Autonomic Dysreflexia. 2001. Paralyzed Veterans of America.

- If the catheter appears to be blocked, gently irrigate the bladder with a small amount of fluid, such as normal saline at body temperature. Avoid manually compressing or tapping on the bladder.
- If the catheter is not draining and the blood pressure remains elevated, remove and replace the catheter.
- Prior to replacing the catheter, instill 2 percent lidocaine jelly (if readily available) into the urethra and wait several minutes.
- If the catheter cannot be replaced, consider attempting to pass a coude catheter, or consult an urologist.
- Monitor the individual's blood pressure during bladder drainage.
- If acute symptoms of autonomic dysreflexia persist, including a sustained elevated blood pressure, suspect fecal impaction.
- If the elevated blood pressure is at or above 150 mm Hg systolic, consider pharmacologic management to reduce the systolic blood pressure without causing hypotension prior to checking for fecal impaction.
- Use an antihypertensive agent with rapid onset and short duration (e.g. nifedipine bite and swallow, 2% nitroglycerin ointment, or prazosin) while the causes of autonomic dysreflexia are being investigated.
- Monitor the individual for symptomatic hypotension.
- If fecal impaction is suspected, check the rectum for stool, using the following procedure: With a gloved hand, instill a topical anesthetic agent such as 2 percent lidocaine jelly generously into the rectum. Wait approximately 5 minutes for sensation in the area to decrease. Then, with a gloved hand, insert a lubricated finger into the rectum and check for the presence of stool. If present, gently remove, if possible. If autonomic dysreflexia becomes worse, stop the manual evacuation. Instill additional topical anesthetic and recheck the rectum for the presence of stool after approximately 20 minutes.
- If precipitating cause of AD is not yet determined, check for other less frequent causes.
- Monitor the individual's symptoms and blood pressure for a least 2 hours after resolution of the autonomic dysreflexia episode to make sure that it does not recur.
- If there is poor response to the retreatment specified above and/or if the cause of the dysreflexia has not been identified, strongly consider admitting the individual to the hospital to be monitored, to maintain pharmacologic control of the blood pressure, and to investigate other causes of the dysreflexia.
- Document the episode in the individual's medical record. This record should include the presenting signs and symptoms and their course, treatment instituted, recordings of blood pressure and pulse, and response to treatment. The effectiveness of the treatment may be evaluated according to the level of outcome criteria reached:
  - The cause of the autonomic dysreflexia episode has been identified.
  - The blood pressure has been restored to normal limits for the individual (usually 90 to 110 systolic mm Hg for a tetraplegic person in the sitting position).
  - The pulse rate has been restored to normal limits.
  - The individual is comfortable, with no signs or symptoms of autonomic dysreflexia, of increased intracranial pressure, or of heart failure.
- Once the individual with spinal cord injury has been stabilized, review the precipitating causes with the individual, members of the individual's family, significant others, and caregivers and provide education as necessary. Individuals with SCI and their caregivers should be able to recognize and treat AD and seek emergency treatment if it is not promptly resolved. It is recommended that an individual with a spinal cord injury be given a written description of treatment for autonomic dysreflexia at the time of discharge that can be referred to in an emergency.

7. **<u>Cardiovascular Effects of Medications in SCI</u>: E.g. Sildenafil (Viagra)**
    - Beware of fatal interaction of Sildenafil with nitrates (e.g. nitropaste for Autonomic Dysreflexia treatment)

8. **<u>Coronary Artery Disease</u>**

- Coronary Artery Disease (CAD) has become an important cause of mortality and morbidity in SCI with increasing survival and aging of the SCI population.
- Specific risk factors for CAD may be increased in SCI. These include – low levels of HDL (high density lipoprotiens), physical inactivity, increased proportion of body fat, and higher incidence of glucose intolerance. The significance of additional emerging CHD risk factors in SCI still needs to be defined.
- **Symptoms and signs**
    - Individuals with SCI above T5 may not perceive chest pain with angina.
    - May present atypically with nausea, unexplained autonomic dysreflexia, jaw pain, episodic dyspnea, syncope, changes in spasticity.
    - On the other hand, GE reflux, which is common in SCI may be mistaken for angina
    - Caution in interpreting signs of cardiac failure (eg limb edema could be due to dependency or CHF, lung crackles could be due to atelectasis vs left heart failure)
- **Diagnostic Evaluation**
    - Traditional treadmill exercise stress test can not be done
    - Arm ergometry can be done in paraplegia. But arm exercise typically produces lower heart rates than leg exercise, and may miss detection of heart disease.
    - Pharmacological stress testing is often the most practical option. Consist of administration of a pharmacological agent such as dipyridamole (Persantin), adenosine, or dobutamine to induce cardiac stress, in combination with a form of cardiac imaging (thallium 201 scanning or 2-D echocardiography).

- **Prevention**
    - **Reduction of cardiac risk factors**. Because of exercise limitations in SCI, reduction of other cardiac risk factors is even more important.
    - Smoking cessation (counseling, behavioral techniques, nicotine patches or gum)
    - Detection and control of hypertension (JNC 7 guidelines)
    - Weight control, diabetic control, lipid management (diet/ drugs - NCEP ATP III guidelines)
    - **Physical Activity/ Exercise in SCI**
        - Activities of daily living alone typically do not result in cardiovascular conditioning. Even modest exercise can achieve health benefits, though may not be able to exercise enough to produce definite cardiovascular benefits with higher levels of injury.
        - **Exercise capacity in SCI** is limited by two main factors: paralysis of the large lower extremity muscles, and the blunted sympathetic response to exercise.
        - Depending on the level of injury, exercise options may include arm-crank ergometry, hand cycling, endurance sports, swimming, Circuit resistance training and electrically induced exercise
    - **Management of Coronary Artery Disease in SCI**
        - Spectrum of interventions available to able-bodied should be offered (Life style changes, medications, angioplasty, CABG)
        - May not be able to tolerate traditional anti-anginal medication doses due to low blood pressure, so introduction of medications may have to be done cautiously

9. Peripheral Vascular Disease

- Significant problem in aging patients with SCI, especially in diabetics and smokers
- Lack of pain and cardinal symptoms of limb ischemia such as intermittent claudication may delay detection of vascular disease. May present in advanced stages with gangrene
- May be contributing to the pathogenesis of non-healing skin ulcers
- Minimizing risk factors such as smoking, diabetes and hyperlipidemia is a key part of management

TABLES

Table 1: ***Cardiovascular Issues in SCI***

Hypotension
Low baseline blood pressure
Orthostatic hypotension
Bradycardia, arrhythmias, cardiac arrest
Autonomic dysreflexia
Reduced cardiovascular fitness and altered exercise capacity
Influence on coronary and peripheral artery disease
Impact on risk factors
Silent ischemia, atypical presentations
Special diagnostic and treatment considerations
Venous thromboembolism
Cardiovascular effects of medications in SCI

Table 2: ***Reversible Factors Exacerbating Symptomatic Postural Hypotension in SCI***

Prolonged recumbency
Rapid position change
Heavy meals
Physical exertion
Hot environment
Dehydration (diarrhea, viral illness)
Sepsis
Medications: Diuretics, antidepressants, alpha blockers, narcotics

Table 3: ***Management of Orthostatic Hypotension in SCI***

Identify and minimize exacerbating factors (Table 2)
Increase salt intake
Compression stockings, abdominal binder
Tilt table, progressive postural challenges
Head-up tilt during sleep
Elevating wheelchair leg rests
Reclining or tilt-in-space wheelchair
Functional electrical stimulation
Biofeedback
? Body-weight supported treadmill training
Pharmacological treatment
Fludrocortisone
Sympathomimetic amines
Ephedrine
Midodrine
Other medications (Little reported use for SCI related OH)

Table 4: ***Symptoms and Signs of Autonomic Dysreflexia***

Sudden, significant increase in blood pressure
Pounding headache
Flushing of the skin above the level of the SCI, or possibly below
Blurred vision, appearance of spots in the patient's visual fields
Nasal congestion
Profuse sweating above the level of the SCI, or possibly below the level
Piloerection or goose bumps above the level of SCI, or possibly below
Bradycardia (may be a relative slowing only and still within normal range)
Cardiac arrhythmias
Feelings of apprehension or anxiety
Minimal or no symptoms, despite a significantly elevated blood pressure

Table 5: ***Potentially Increased Cardiovascular Risk Factors after SCI***

Decreased physical activity
Low HDL cholesterol
Impaired glucose tolerance, insulin resistance
Increased proportion of body fat
Psychosocial factors (Depression, Social isolation)
Hypothesized effects of SCI on emerging risk factors

Table 6: ***Key Prevention Targets for Coronary Heart Disease (CHD)***

Smoking cessation
Lipid management to goal
Blood pressure control
Weight management
Physical activity
Diabetic management to goal
Additional components of secondary prevention with known CHD
Antiplatelet agents (aspirin), anticoagulants
Renin-angiotensin aldosterone system blockers
Beta blockers

Table 7: ***Unique Issues in the Diagnosis of CHD after SCI***

Atypical presentations, lack of chest pain
Under-diagnosis of coronary heart disease (CHD)
Delayed treatment; Inadequate secondary prevention
Confusing physical signs
Dependent edema versus heart failure
Atelectasis versus left ventricular failure
Nonspecific ST-segment and T-wave changes in SCI
Cardiac stress testing
Inability to perform traditional treadmill test
Suboptimal sensitivity of arm versus leg exercise
Difficulty in interpreting significance of exercise induced hypotension
Indication for pharmacologic stress testing

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